<Case Report>A Case of Internal Carotid Artery Occlusion Complicating Embolization of the External Carotid Artery System

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CITATION:

YAMAGAMI, TATSUHITO ...[et al]. <Case Report>A Case of Internal Carotid Artery Occlusion Complicating Embolization of the External Carotid Artery System. 日本外科宝函 1997, 66(2): 51-58

ISSUE DATE:

1997-05-01

URL:

http://hdl.handle.net/2433/202868

Arch Jpn Chir 66(2), 51～58, Mai., 1997

症　　例

A Case of Internal Carotid Artery Occlusion Complicating Embolization of the External Carotid Artery System

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Received for publication, Apr. 27., 1997

Abstract

A case of visual disturbance by embolization through the external carotid artery (ECA) after internal carotid artery (ICA) occlusion is reported. A 65-year-old female presented left ICA occlusion 14 years ago. She was admitted with acute deterioration of left visual acuity. Ophthalmologically, the retinal central artery was occluded.

Cerebral angiography disclosed an irregular wall at the left ICA stump and stenosis of the maxillary artery. Carotid endarterectomy of the common carotid artery, ECA and residual ICA was performed and atheromatous plaque with ulceration was removed. The postoperative course was uneventful. We discuss cases of ICA occlusion in relation to visual impairment.

Introduction

Carotid artery diseases are responsible for 20 to 30% of cerebral infarct in the United States. In Japan this tendency is increasing.

Ischemic changes of the eye and cerebral hemisphere are common in ipsilateral carotid artery stenosis or occlusion at its origin. Arterial embolus is apt to occur in ulcerative lesions. Microembolism of the ophthalmic artery typically causes amaurosis fugax. Among carotid artery diseases visual impairment can occur after embolization through the external carotid artery system. We report on such a case.

Case Report

A 65-year-old female noticed transient motor weakness of the right upper limb three times between 1979 and 1980.

In April of 1982, vomiting, dysarthria and left hemiparesis occurred. CT disclosed a small low density area from the right internal capsule to the right corona radiata. Cerebral angiography re-

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索引用語：内頸動脈閉塞，塞栓，外頸動脈，視力障害
Key words: Internal carotid artery occlusion, Stump, Embolization, External carotid artery, Visual disturbance

vealed stenosis of the right middle cerebral artery (MCA) and occlusion of the left internal carotid artery (ICA). After superficial temporal artery – middle cerebral artery (STA-MCA) anastomosis of the right side, she became asymptomatic and received antiplatelet therapy.

In May 1996, nausea was experienced in the early morning. She noticed a cold and heavy sensation of the left lower limb. Follow-up MR angiography (MRA) and cerebral angiography showed that STA-MCA bypass of the right side was patent and that left ICA was occluded at its origin. On July 25, 1996, her left visual acuity decreased in the early morning. Visual field loss rapidly increased throughout the day and she consulted us in the evening.

She was alert and cooperative. Initial examination disclosed nearly loss of left visual acuity in counting fingers at 30 cm. Residual visual field of ths left eye was only a small portion lateral to the middle. She noticed numbness of the left half of the tongue. Motor activity of upper and lower limbs was good.

CT on admission revealed a small low density area from the right lentiform nucleus to the right corona radiata (Fig. 1). It was essentially the same as that presented 14 years previously. MRI showed ischemic lesion of the left thalamus in addition to that of CT findings (Fig. 2).

On MRA, left ICA, left ophthalmic artery and left MCA were not visualized (Fig. 3). Right MCA showed stenosis at the M1 portion. MRA of the cervical portion showed occlusion of the left ICA at its origin (Fig. 4).

Left carotid angiography (CAG) revealed occlusion of the left ICA at its origin (Fig. 5) and an irregular wall at its residual stump (Fig. 6). Irregular wall of the maxillary artery was also demonstrated (Fig. 6). Right CAG showed stenosis of the right MCA, tha same finding as that of MRA (Fig. 7). Collateral flow through the anterior communicating artery was not visualized. Collateral flow through the posterior communicating artery on vertebral angiography was not visualized, either.

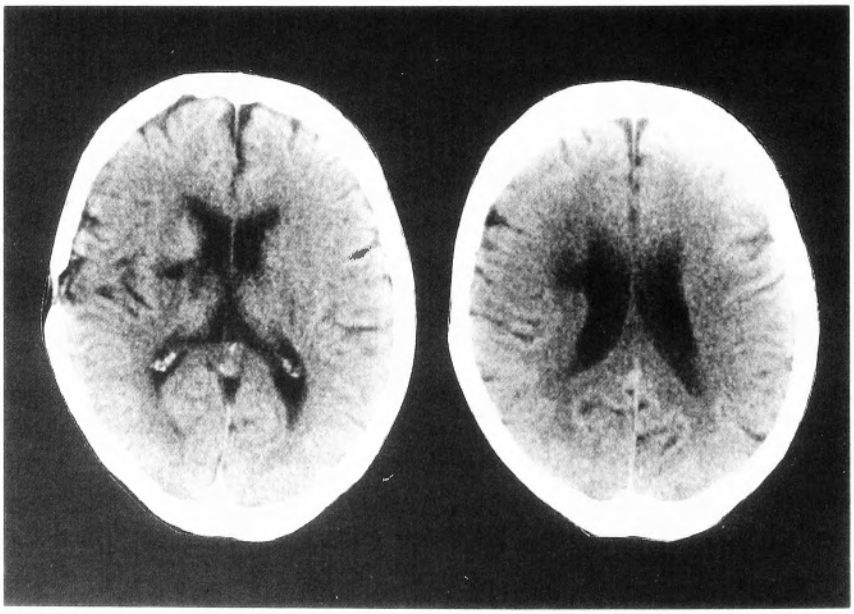

Fig. 1 CT disclosed low density area of the right lentiform nucleus and right corona radiata.

Drip infusion that included urokinase was started. Ophthalmic examination showed obstruction of the left retinal central artery. The embolus at the left ICA stump was thought to flow into the retinal central artery via the external carotid artery (ECA) system. Carotid endarterectomy (CEA) from the left common carotid artery (CCA) to the left ICA and ECA was performed. Atheromatous plaque with ulceration was removed.

Single photon emission CT showed decreased cerebral blood flow of the left frontal and temporal lobes. After four weeks, left STA-MCA anastomosis was performed. The postoperative course was uneventful. Left visual acuity was unchanged.

Discussion

Symptoms of embolic events can be derived from atheromatous ulceration or thrombus formation in the ECA, the CCA or the stump or cul-de-sac at the origin of the occluded ICA. These symptoms are usually ipsilateral amaurosis fugax and lateralizing hemispheric transient ischemic attacks caused by cerebral hypoperfusion or embolization. Visual deterioration of nearly blind state rarely

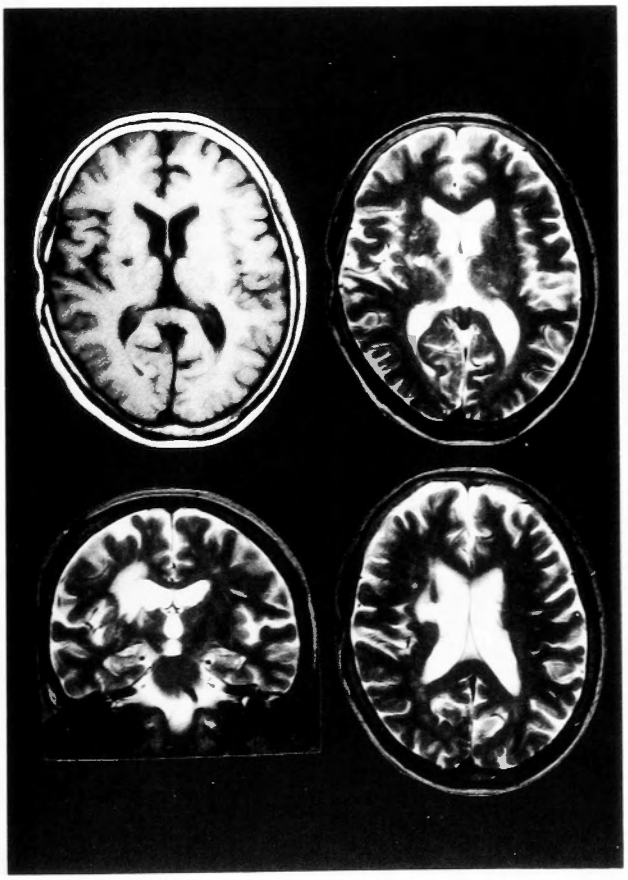

Fig. 2 MRI showed ischemic lesion of the left thalamus in addition to the right lentiform nucleus and right corona radiata. Axial T1 weighted image (upper left), axial T2 weighted (T2W) images (upper right and lower right) and coronal T2W (lower left) image.

occurs. In the present case, microembolism moving from the distal stump of occluded ICA caused ischemia of the retinal central artery through ECA, the maxillary artery and the ophthalmic artery via the periorbital collateral pathway.

BARNETT et al. reported that the stump of ICA should be regarded as an important source of fur-

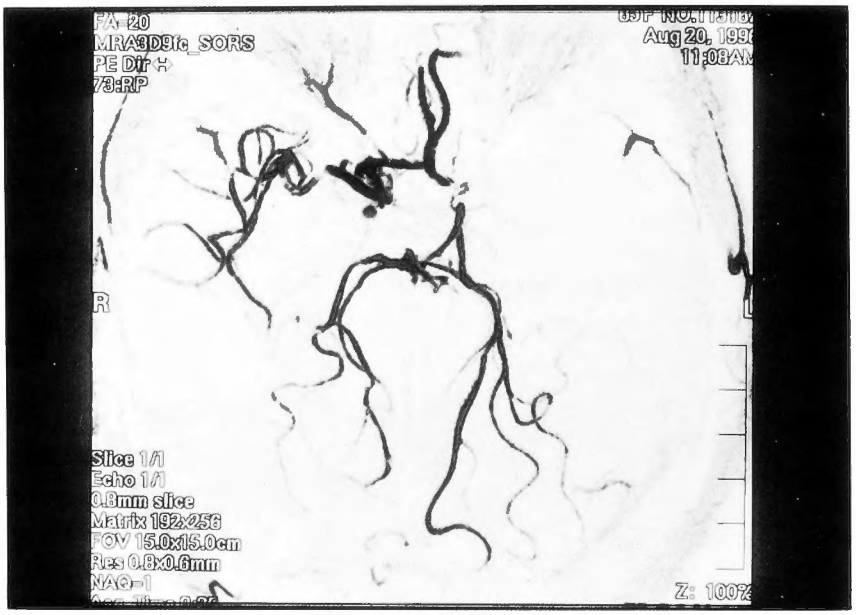

Fig. 3 Cranial MRA showed absence of distal ICA, ophthalmic artery and MCA of the left side.

Fig. 4 Cervical MRA showed occlusion of the left ICA at its origin.

ther cerebral embolic ischemia[1]. They suggested that turbulence in the stump contributed to progressive atherosclerotic changes and aggravated thrombogenesis in addition to platelet aggregation and endothelial damage. Thromboembolism from the stump via the anastomotic supply through ipsilateral CCA and ECA is thought to be responsible for ischemic events to the brain or retina.

TERAO et al. analyzed nineteen cases of amaurosis fugax[2]. They reported that microembolism was derived via the ECA in five cases. There are many other pathologic processes that might underlie amaurosis fugax in Japan, besides microembolus originating from the origin of the ICA whose etiologic importance has been emphasized in Western countries. They referred to the necessity of evaluation of blood flow in the ophthalmic artery by transcranial Doppler method.

TAKAHASHI et al. summarized the pathogenesis of transient monocular blindness[3]. These include: 1) Embolism-thromboembolism at the bifurcation of CCA, atheromatous embolism of the innominate artery or aortic arch and distal ICA, cardiogenic embolism (valvular thrombus, mural thrombus, atrial myxoma), embolus as described in intravenous drug abusers, 2) Hemodynamic change-widespread atheromatous occlusive vascular disease, Takayasu's arteritis, hypoperfusional state (heart failure, acute decrease of circulating blood volume, polycythemia, thrombocytosis, hypercoagulative state as a consequence of cancer or pregnancy) 3) Ophthalmic disease-anterior ischemic optic neuropathy, retinal central artery occlusion, obstruction of the retinal central vein, non-vascular origin (elevated intraocular pressure or ocular compression, periorbital trauma) 3) Neurologic disease-disorder of the brain stem, vestibular system or oculomotor nerve system, optic neuritis, opticochiasmal compressive lesion, papilledema, multiple sclerosis, migraine 4) Idiopathic origin

Generally, patients, of ICA occlusion may be observed without any special treatment. The present case has clinically been asymptomatic for fourteen years. WAYBRIGHT *et al. states that visual loss occurs in 10% of individuals with ipsilateral ICA occlusion*[4]. *Rich intraorbital collateral system may compensate for*

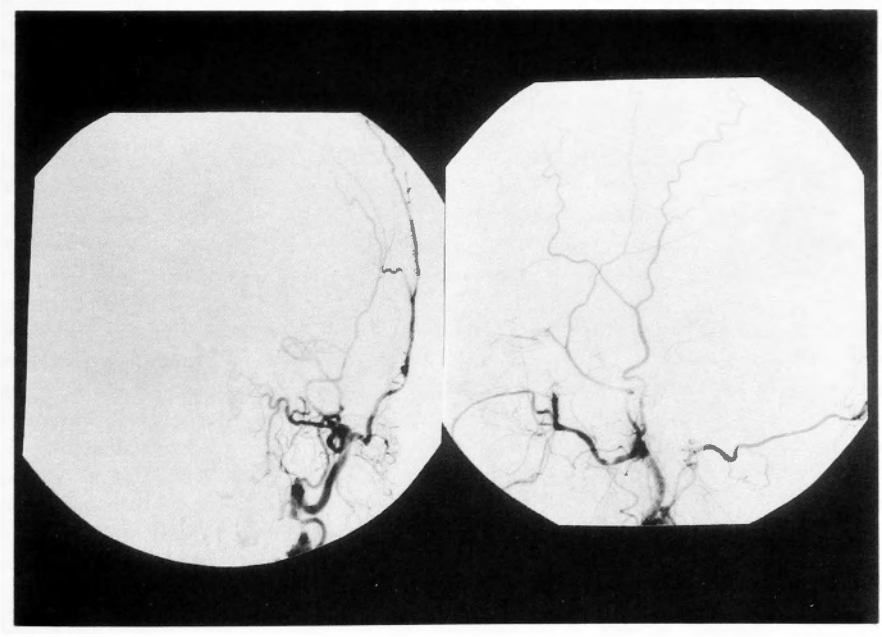

Fig. 5 Left CAG showed occlusion of the left ICA. Only branches of the ECA were visualized. Anteroposterior (AP) view (left) and lateral view (right).

the appearance of neurologic symptoms after ICA occlusion[5]. *But* BOGOUSSLAVSKY et al. reported a case of acute orbital infarction[6].

DOUGLAS et al. concluded that embolism was the suspected etiology in 73% of retinal central

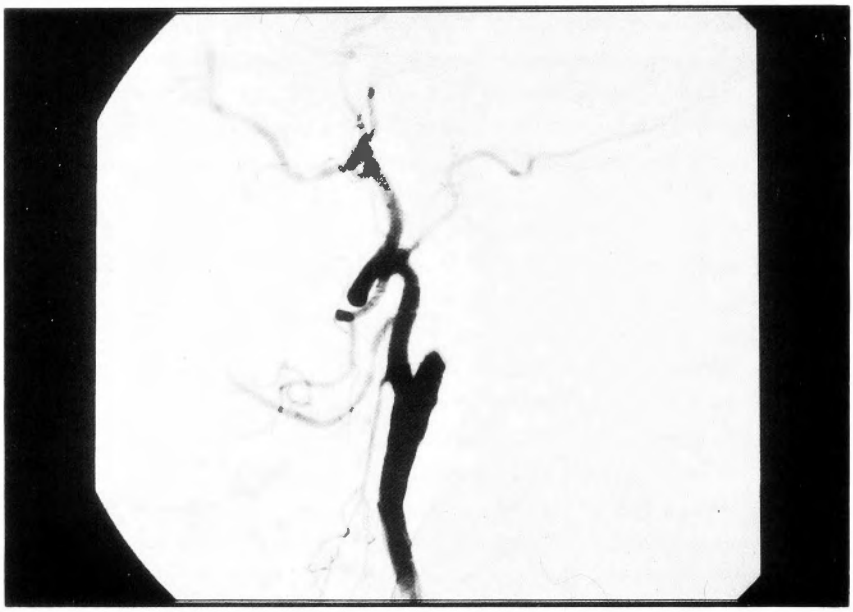

Fig. 6 Left ICA was occluded at its origin. Internal wall of the CCA and ICA was irregular. Stenosis of maxillary artery was also found.

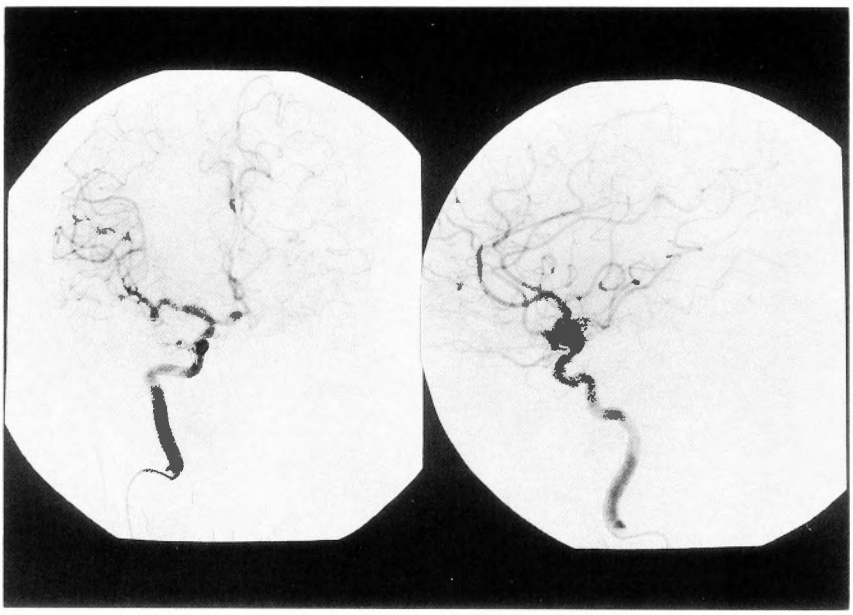

Fig. 7 Right CAG showed mild stenosis of the right MCA and carotid siphon. AP view (left) and lateral view (right).

artery occlusions[7]. There is higher incidence of subsequent ipsilateral stroke in retinal central artery occlusion patients with carotid artery disease. They recommended CEA in retinal central artery occlusion patients who were found to have associated carotid artery disease.

In this respect, endarterectomy of the stenotic ECA associated with ipsilateral occluded ICA is of importance[5,8–10]. The benefits of CEA have been evaluated in symptomatic patients with high grade stenotic lesions of ICA origin and ulcerative stenotic lesions to reduce the incidence of cerebral infarction[11–14].

Operative treatment of the stump at the origin of the occluded ICA usually involves stumpectomy[5,9,10,12,15,16]. Endarterectomy of CCA and ECA with transection or closure of the residual ICA stump are performed to prevent future embolic events occurring from the residual orifice. However, part of the ICA cul-de-sac with smooth intima was residual in our patient. Postoperative CAG confirmed the smooth wall of the residual ICA. Embolization from the residual ICA stump may enter the cerebral hemisphere in the future. Follow-up examinations must be thorough and stumpectomy at the appropriate period will have to be considered.

In occlusive disease of ICA origin, deterioration of visual symptoms can be prevented if removal of the embolic source to prevent entry to the ophthalmic artery system is performed when patients complain of mild or transient visual disturbance.

Acknowledgement

We thank Asayo Numa, M.D. (Numa Ophthalmic Clinic, Joyo) for her careful ophthalmic examination.

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和文抄録

外頸動脈系の塞栓が生じた内頸動脈閉塞の1例

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浦西龍之介

内頸動脈閉塞の経過観察中に外頸動脈を介して視力障害を呈した1例を経験したので報告する．

【症例】65歳，女性．14年前に左内頸動脈閉塞と右中大脳動脈狭窄を認め，右側の浅側頭動脈中大脳動脈皮質枝吻合術 (STA-MCA anastomosis) を受けた．左視力障害が急速に進行した．来院時にはほぼ失明に近い状態で，眼科的には，網膜中心動脈の閉塞であった．左内頸動脈起始部閉塞部の stump に，ulcer を伴う atheromatous plaque があり，外頸動脈系を介し，網膜中心動脈へ embolism が生じたと診断された．

Carotid endarterectomy と左側の STA-MCA anastomosis を施行し，術後経過は順調であった．内頸動脈閉塞例で，外頸動脈，総頸動脈，あるいは閉塞した内頸動脈起始部での stump あるいは cul-de-sac からの embolism により，amaurosis fugax や大脳半球の虚血症状が生ずることはよく認められているが，失明かそれに近い状態になることは稀である．軽度あるいは一過性の視力障害のうちに，眼動脈系への embolic source を取り除けば，高度の視力障害は未然に防ぐことができると考えられる．